comment in an otherwise sensitive strain would cause doubt and confusion. It is necessary, therefore, to state that the strain has the cultural characters of *M bovis*. Some strains of *M bovis* also show a low level of resistance to isoniazid. This is not detrimental to the use of the drug but again would require a comment.

The "others"—Again, we are agreed that no single collective noun is universally acceptable for those mycobacteria that are not tubercle bacilli. To call them "anonymous," however, because at the moment of isolation they are unidentified is also unscientific. All bacteria at the moment of isolation could be deemed anonymous because various tests have to be undertaken before a particular strain is identified. Any laboratory with a modicum of experience should be able in a few days to issue an interim report as to the likely identity of a mycobacterium. This is particularly so if it belongs to a species which is likely to be pathogenic, and an answer is rarely required more quickly. The form of words used to convey the likelihood of a particular isolate being clinically important is not important so long as the physician understands the report. To suggest, however, that every report of the isolation of *M gordonae* or *M terrae* be qualified because such organisms may infect immunocompromised subjects is excessive. Can the authors produce evidence as to the incidence of such infections?

The authors also neglect to mention the importance of the specimen from which an opportunist mycobacterium is isolated. In urine or gastric washings they are virtually never important, in sputum they may be and further isolates should be sought, in tissues and aspirates they should be but can sometimes still enter the specimen accidentally.

The tone of the article is patronising in its stated aim of protecting the physician from the niceties of nomenclature. It is one thing to take the identification of an organism only as far as is clinically necessary and to provide the physician with clear advice as to its likely clinical importance. It is wrong to do this by adding to confusion over nomenclature.

P A JENKINS

PHLS Mycobacterium Reference Unit,
Heath Park,
Cardiff CF4 4XW

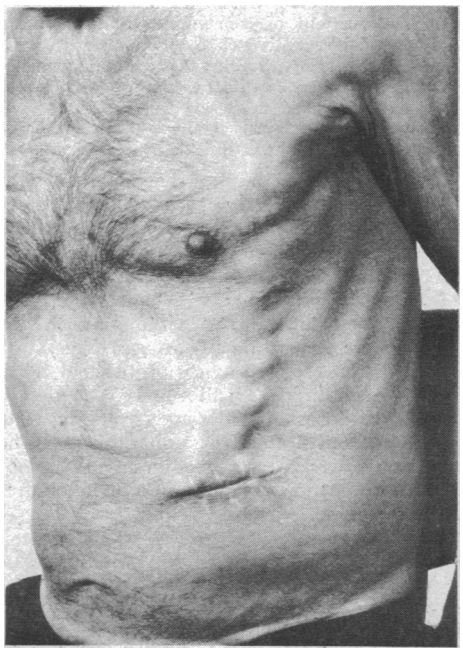
Development of tumour along the track of a peritoneovenous shunt

SIR,—Dr D Tarin and others (10 March, p 749) found that no clinically important metastases developed in their patients with peritoneovenous shunts. We report a case where troublesome tumour nodules developed along the subcutaneous track of a failed shunt.

A 61 year old man presented in August 1983 with a one month history of abdominal swelling, back pain, anorexia, and weight loss. Examination showed gross ascites and an irregular epigastric mass. The ascites was drained, and cytological examination showed many poorly differentiated adenocarcinoma cells. Investigations showed haemoglobin 12·5 g/dl, a white cell count of $8·3 \times 10^9$/l (62% neutrophils, 17% lymphocytes, 14% monocytes, 6% eosinophils, and 1% basophils), and platelets 748×10^9/l. Prothrombin time was 17·5 s (control 11 s). There was no evidence of disseminated intravascular coagulation. Liver function tests and serum amylase concentration were normal. Chest *x* ray and barium meal examinations showed no abnormality.

On 18 August a LeVeen shunt was inserted into the left upper quadrant of the abdomen and the left external jugular vein via a subcutaneous tunnel. After functioning for a few weeks there was reaccumulation of ascites, and the shunt was removed on 13 October. Examination of the shunt showed blockage of the valve with tumour. The ascites was drained and 60 mg of bleomycin was instilled into the peritoneum.

In January 1984 he complained of abdominal discomfort and of multiple tender nodules along the track of the shunt. On examination, there was a large mass in the upper abdomen and tender nodules along the subcutaneous track of the shunt, (see figure). There was no evidence of distant



Development of tumour nodules along the track of a peritoneovenous shunt.

metastases. He was started on chemotherapy, a combination of fluorouracil and mitomycin C, with some relief of his discomfort. When last seen on 18 March there was no change in the size of the abdominal tumour or the subcutaneous nodules and no evidence of ascites.

To the best of our knowledge, this complication of peritoneovenous shunts has not been described.

G G KHOURY
D J T WEBSTER
C W KEEN

Combined Breast Clinic,
Velindre Hospital,
Cardiff CF4 7XL

Endotracheal cuff pressure and tracheal mucosal blood flow: endoscopic study of effects of four large volume cuffs

SIR,—I read with interest the article by Dr R D Seegobin and Dr G L van Hasselt (31 March, p 965) on the effects of large volume endotracheal cuffs on tracheal blood flow. A study of two types of endotracheal tubes, one a high pressure low volume type (Portex Blueline), the other a low pressure high volume type (Lanz), has shown less damage to the tracheal mucosa when assessed endoscopically during extubation 24 hours after cardiac surgery.[1] The bronchoscopist scored the degree of tracheal damage seen and also photographed the trachea, and later an independent observer scored the degree of damage seen on the photographs. Both observers found significantly less ($p < 0·01$ and $p < 0·05$ respectively) damage caused by the Lanz low pressure endotracheal tube.

This suggests that Seegobin and van Hasselt's observations are relevant as early as 24 hours postoperatively and, as they comment, it seems the tracheal lateral wall pressure is the most important factor causing this damage. It seems that this pressure should not exceed 30 cmH_2O and so the Lanz tube's cuff pressure of 27 ± 2 cmH_2O is suitable. Our patients who were randomly allocated to the Lanz tubes did not, however, escape tracheal damage completely—so it seems that capillary perfusion pressure is being exceeded in some patients even at this low cuff pressure. But lowering the cuff pressure even further may lead to an increased number of patients having an inadequate clinical seal.

D HONEYBOURNE

Hope Hospital,
Salford M6 8HD

[1] Honeybourne D, Costello JF, Barham C. Tracheal damage after endotracheal intubation: comparison of two types of endotracheal tubes. *Thorax* 1982; **37**:500-2.

SIR,—While agreeing with Dr R D Seegobin and Dr G L van Hasselt that cuff inflation technique is the most important prerequisite to avoiding tracheal mucosal damage (31 March, p 965) we think that the reader might be left with the impression that 25 cmH_2O is an acceptable inflation pressure to use in all cases. We want to emphasise that care should be taken when inflating the cuff to aim for a pressure just providing a cuff seal and no further. This will help to minimise the effects of variation in blood pressure and the diffusion of nitrous oxide into the cuff.

P ARCHER
J BROOKER

Department of Anaesthesia,
Royal Victoria Infirmary,
Newcastle upon Tyne NE1 4LP

Oxygen as a driving gas for nebulisers: safe or dangerous?

SIR,—We share with Dr H Cass and others (31 March, p 1009) the worry that salbutamol nebulisation with air may aggravate hypoxaemia, although this was not evident from our study (28 January, p 272). Dr Cass and others write that the fall in arterial oxygen pressure that can occur when nebulisers are driven with air is well described, but we could not find a well designed study which specifically investigated this point. Nobody has documented the degree and duration of hypoxaemia that occurs with nebulised salbutamol in different categories of patients with airways obstruction and its relation if any to the severity and the reversibility of the airway obstruction.

We have data for 29 patients with chronic bronchitis and emphysema who had serial blood gas measurements during and up to 30 minutes after 5 mg of salbutamol was delivered by an air driven nebuliser. Each patient had at least eight arterial blood samples taken at regular intervals through an arterial cannula. Their mean baseline arterial oxygen pressure was 8·1 kPa (60·6 mm Hg) (SD 1·84 kPa (13·1 mm Hg); range 4-11·7 kPa (30-88 mm Hg)). There was no consistent pattern to the changes in the arterial oxygen pressure during or after nebulised salbutamol. If one ignored the fluctuations due to experimental error and took only the maximum observed fall in oxygen pressure, then 17 patients had a fall of less